

SHORT REPORT

Molluscum contagiosum surrounded by a white halo and Sezary syndrome

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Abstract

Background Molluscum contagiosum (MC) is caused by a DNA virus of the poxvirus group. It is common in children, and is also found in sexually active adults and HIV-seropositive patients. Cellular immunity is essential to controlling MC virus infection. We report the first observation of a patient with stage IV Sezary syndrome, who presented multiple molluscum contagiosum, spread and surrounded by a pale halo.

Case report A woman aged 70 presented with aggravation of Sezary syndrome diagnosed in 2009 and treated with topical corticosteroids. The examination showed a generalized pruritic exanthem and multiple flesh-coloured papules from 1 to 3 mm, spread over the entire skin surface and surrounded by a white halo. Histological examination of a lesion showed the presence of infected cells with intracytoplasmic inclusions infected in an acanthotic epidermis, surrounded by a melaninopenic hypomelanosis with a normal melanocyte density. There was no inflammatory character. The diagnosis of multiple molluscum contagiosum was given, the application of clobetasol propionate was suspended and treatment with chlorambucil 4 mg/day and prednisone 0.5 mg/kg/day was started. The evolution of the rash and pruritus was rapidly favourable. After 3 months, the rash and pruritus had regressed. There was no molluscum contagiosum or clear halo.

Conclusion We report the original observation of a patient with stage IV Sezary syndrome, who presented multiple molluscum contagiosum, spread and surrounded by a pale halo, without inflammation, eczema or disappearance of melanocytes. This halo could be due to the secretion of a protein by molluscum contagiosum inhibiting inflammation around this MC. To our knowledge, this phenomenon reported in a patient with severe atopic dermatitis associated with Sezary syndrome has not previously been described.

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Conflicts of interest

None.

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Introduction

Molluscum contagiosum (MC) is caused by a DNA virus of the poxvirus group. It is common in children and also observed in sexually active adults and HIV-seropositive patients. Cellular immunity is essential to controlling MC Virus (MCV) infection. We report the first observation of a patient with stage IV Sezary syndrome, who presented multiple molluscum contagiosum, spread and surrounded by a pale halo.

Case report

A woman aged 70 years presented with aggravation of Sezary syndrome, diagnosed in 2009. Initially, blood immunophenotyping showed lymphomatous infiltration T in 57% of total lymphocytes. The whole body scan was unremarkable and the myelogram revealed the presence of rare cells suggestive of Sez-

ary cells. Treatment with topical corticosteroid and simple monitoring were decided on based on the asymptomatic nature. In October 2012, a generalized pruritic exanthem arose, which prompted a skin biopsy. Histological analysis confirmed Sezary syndrome through the presence of a dermoepidermal lymphocytic infiltrate having an epidermotropism with the formation of Pautrier's microabscesses. The immunohistochemical examination confirmed a phenotype T CD3+ CD4+ CD8-. The blood count showed leukocytosis at 18.570 G/L with lymphocytosis at 12.999 G/L and the presence of 20% circulating Sezary cells (3.710 G/L). The ratio of CD4 to CD8 lymphocytes was 90.99. The myelogram showed the presence of 27% of Sezary cells. No abnormality was found in the scanner. Clinical examination revealed a generalized pruritic exanthem and multiple flesh-coloured papules from 1 to 3 mm, spread over the entire skin sur-



Figure 1 Exanthem with multiple flesh-coloured papules, surrounded by a clear halo.



Figure 2 Hemispherical papule with umbilical centre.

face and surrounded by a white halo (Fig. 1). The papules were hemispherical with an inverted centre (Fig. 2). According to the patient, moles had been present since childhood, multiplying over the years. The clear halo was recent. The histological examination of a lesion showed the presence of infected cells with intracytoplasmic inclusions in an acanthotic epidermis (Fig. 3), surrounded by a melaninopenic hypomelanosis (Fontana) with a normal melanocyte density after MelanA staining. There was no inflammatory character. The diagnosis of

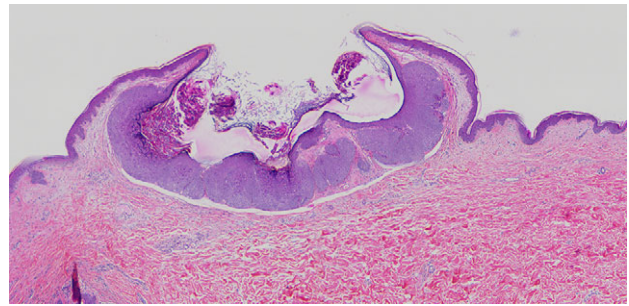


Figure 3 Molluscum contagiosum. Acanthotic epidermis, invaginated into piriform lobules, aggregation of viral particles.

molluscum contagiosum multiple was given, the application of clobetasol propionate was suspended and a sedative treatment with hydroxyzine dihydrochloride was introduced. Regarding stage IVB Sezary syndrome, treatment with chlorambucil 4 mg/day and prednisone 0.5 mg/kg/day was started. The evolution of the exanthem and pruritus was rapidly favourable. After 3 months, the exanthem and pruritus had regressed. There was no more molluscum contagiosum or clear halo. Chlorambucil treatment was continued associated with a gradual decrease of corticosteroids.

Discussion

The MCV is caused by a DNA virus of the poxvirus group.¹ It is common in children, due to human transmission or self-inoculation by scratching. It can occur anywhere on the body. Its spread is often significant in atopic children,² favoured by pruritus and abnormal cellular immunity. It is also found in sexually active adults and in HIV-seropositive patients.^{3–5} MCV infection affects 9% of patients infected with HIV.⁶ Cellular immunity is essential to controlling MCV infection.⁷ Th2 dysregulation of the immune system, found in atopic patients and HIV-seropositive patients,⁸ plays a supporting role by lowering defences and controlling infection.⁹ Cases of MCV infections have also been reported in patients treated with chemotherapy¹⁰ or corticosteroids.⁷ Several cases of disseminated MCV infection were reported in patients treated with methotrexate,^{10–12} including a case recently reported by Bansal *et al.*¹² Methotrexate causes a reduction in TNF α and INF γ , cytokines allowing the apoptosis of infected cells and blocking viral transcription.¹¹ MCV infections have also been described in patients with mycosis fungoides.^{13,14} Our patient had a profuse shape with spread molluscum contagiosum, which can be explained by cellular immunity anomalies induced by Sezary syndrome, T lymphoproliferation with Th2 expression,¹⁵ such as mycosis fungoides.¹⁶ To our knowledge, no cases of spread MC associated with Sezary syndrome have been reported in the literature.

The second feature of our observation is the presence of a white halo around each MC. The inflammatory reactions around

the MC, common in children,¹⁷ are characterized by an erythema, an oedema and sometimes an eczema around the MC called molluscum dermatitis.¹⁸ Eczematized molluscum are more frequent in paediatric patients with atopic dermatitis (AD) and pruritus causes a larger number of MC.¹⁹ The inflammation of MC usually involves the disappearance of lesions within a few months.¹⁷ An inflammatory infiltrate with many lymphocytes is highlighted on the histological images.¹⁸ The patient showed no eczematized molluscum or inflammatory appearance on the histological images.

Pastin²⁰ reported a case of MCV surrounded by a clear area, in a patient with an AD and severe eczema. According to the author, this clear zone may be due to inhibition of AD by the MCV.²⁰ A protein, MC148, secreted by the MCV prevents the expression of AD around the molluscum. This protein, by binding to its CCR8 receptor, inhibits inflammation of AD and may also inhibit other Th2-type cell-mediated inflammatory reactions, expressing CCR8.²⁰

Our patient had no AD, however, she did have an erythroderma by Sezary cell infiltration. According to Fiero *et al.*,¹⁵ Sezary syndrome involves a type Th2 inflammatory response, and therefore suggests that the MCV may inhibit Sezary syndrome by secretion of an inhibitory protein.

Another phenomenon could be closer; Woronoff's ring described in 1926 and characterized by a pale halo of healthy skin around the psoriasis plaques. Its pathophysiology is not completely understood. Penneys *et al.*²¹ suggested the presence of a prostaglandin synthesis inhibitor, which comes from the psoriasis plaque and spreads around it and inhibits the inflammatory reaction. Van de Kerkhof *et al.*⁶ showed a decrease in endoglin levels in connection with a reduction of local inflammation. The histological characteristics of this ring are defined by a minimal epidermal acanthosis and a decrease in the amount of melanin.²²

The histological examination of the patient also shows a melaninopenic hypomelanosis with a very low pigment density localized primarily in the melanocytes. The melanocyte population in the ring is relatively dense.

Sutton's phenomenon, a depigmentation halo surrounding one or more nevi, characterized histologically by very dense lymphocytic inflammatory infiltrates and the disappearance of melanocytes in the white macule can therefore be rejected. The pathogenic mechanism could correspond to an immunological reaction, resulting in the destruction of naevus cells and total repigmentation of the area.²³

Conclusions

We report the original observation of a patient with stage IV Sezary syndrome, who presented multiple molluscum contagiosum, spread and surrounded by a pale halo, without inflammation, eczematization or disappearance of melanocytes. This halo could be due to the secretion of a protein by the molluscum inhibiting inflammation around this MC. To our knowledge,

this phenomenon reported in a patient with severe AD associated with Sezary syndrome has not previously been described.

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